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Emergent Coronary Angioplasty in the Treatment of Acute Ischemic Mitral Regurgitation: Long-Term Results in Five Cases

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Severe mitral regurgitation in the setting of an evolving myocardial infarction is associated with a high operative mortality rate. Five patients with acute severe mitral regurgitation secondary to ischemic posterior papillary muscle dysfunction underwent emergent percutaneous transluminal coronary angioplasty. Two patients were in cardiogenic shock and required intraaortic balloon counterpulsation. Angioplasty resulted in rapid improvement in hemodynamic variables, and all patients were discharged at a mean of 10 days after the procedure.

Long-term follow-up study (mean 35 ± 6 months) revealed normal mitral valve function angiographically and by Doppler echocardiography in four patients. Repeat angioplasty was required in one patient, and another underwent coronary artery bypass surgery without valve replacement for restenosis. One patient developed progressive mitral regurgitation and required elective mitral valve replacement 12 months after angioplasty.

These preliminary findings suggest that emergent coronary angioplasty is a useful therapeutic intervention in the treatment of ischemic mitral regurgitation and is associated with a favorable long-term outcome.

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The occurrence of significant mitral regurgitation in the setting of an evolving myocardial infarction usually results from severe ischemic dysfunction of the posterior papillary muscle (1–3). Restoration of blood flow in this setting is mandatory to prevent acute papillary muscle rupture or fibrosis with subsequent chronic mitral regurgitation (4,5). Because most cases of ischemic papillary muscle dysfunction are not associated with significant left ventricular dysfunction, correction of the hemodynamic lesion would be associated with a favorable long-term survival (4).

Surgical treatment of ischemic mitral regurgitation often requires both valve replacement and coronary artery bypass surgery and is associated with a high mortality rate (6–10). Since its introduction in 1977, percutaneous transluminal coronary angioplasty has been successfully applied to a large subset of patients with ischemic heart syndromes including unstable angina, multivessel coronary artery disease and acute myocardial infarction with and without cardiogenic shock (11–15). In this report, we describe five patients with acute severe mitral regurgitation secondary to ischemic papillary muscle dysfunction who underwent emergent coronary angioplasty of the culprit lesion. Sustained long-term benefit was obtained in four patients; one patient required elective mitral valve replacement for progressive mitral regurgitation 12 months after angioplasty.

Methods

Study patients (Table 1). From June 1982 to January 1986, 150 patients underwent emergent percutaneous transluminal coronary angioplasty at Washington Adventist Hospital for the treatment of evolving myocardial infarction. Five patients had evidence of moderate or severe acute mitral regurgitation secondary to ischemic papillary muscle dysfunction and they form the basis of this report. Their ages ranged from 43 to 82 years (mean 59); there were two men and three women. All patients manifested radiographic evidence of pulmonary edema on presentation (Fig. 1). All patients were in New York Heart Association functional

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Table 1. Clinical and Biochemical Features in Patients With Ischemic Mitral Regurgitation

Patient No.	Age (yr) & Gender	Infarct Location	Interval From Chest Pain to PTCA (h)	Pulmonary Edema	Peak Cpk (IU/liter)	Comments
1	53M	Inferior	4.0	Yes	1,226	—
2	43F	Inferior	8.0	Yes	5,286	Intubated, IABP, heart block, streptokinase
3	82F	Posterior	1.5	Yes	2,344	Intubated, IABP, heart block, streptokinase
4	52M	Inferior	3.0	Yes	4,426	Streptokinase
5	65F	Inferior	2.5	Yes	1,226	Chronic renal failure

Cpk = creatine kinase; F = female; IABP = Intraaortic balloon pump; M = male; PTCA = percutaneous transluminal coronary angioplasty.

class III or IV. Two patients were in cardiogenic shock; both had had cardiopulmonary arrest and required intubation and intraaortic balloon counterpulsation.

Catheterization and angioplasty procedure. Right and left heart catheterization was performed by the femoral approach. Right heart hemodynamic data were obtained with a Swan-Ganz catheter, and cardiac output was determined by the thermodilution technique. Left ventriculography was performed in the 30° right anterior oblique projection to assess left ventricular function and to semiquantitate the degree of mitral regurgitation. Mitral regurgitation was graded 0 to 4+ according to the criteria of Fuchs et al. (16). Angiography of both coronary vessels was performed in multiple views with and without angulation. Percent luminal narrowing was determined manually from at least two views with use of a caliper method before and after angioplasty.

Coronary angioplasty was performed with the technique described by Gruentzig et al. (11), utilizing a fixed or steerable guide wire system. Before insertion of the guide catheter, all patients received 10,000 U of intravenous heparin. Three patients were given 25,000 to 50,000 U of intracoronary streptokinase during the angioplasty procedure. Right heart hemodynamic measurements were repeated 10 to 20 min after angioplasty, and the patients were transferred to the coronary care unit. Intravenous heparin at a mean dose of 1,000 U/h was continued as a continuous infusion for at least 24 h, after which vascular sheaths were removed.

Results

Angioplasty results. Angiographic and hemodynamic variables are detailed in Table 2. Total or subtotal occlusion involved a dominant left circumflex artery in three patients and a dominant right coronary artery in two. Angioplasty was performed 1.5 to 8 h (mean 3.8) after the onset of chest pain and was successful in all patients, resulting in a reduction in percent luminal narrowing from a mean of 99% to 30% (Fig. 2). Hemodynamic measurements made before and 10 to 20 min after successful angioplasty demonstrated a marked

Figure 1. Patient 1. Chest radiographs before (**A**) and 2 days after (**B**) angioplasty. Note resolution of cardiomegaly and absence of bilateral hilar infiltrates and pulmonary venous hypertension after angioplasty (**B**).

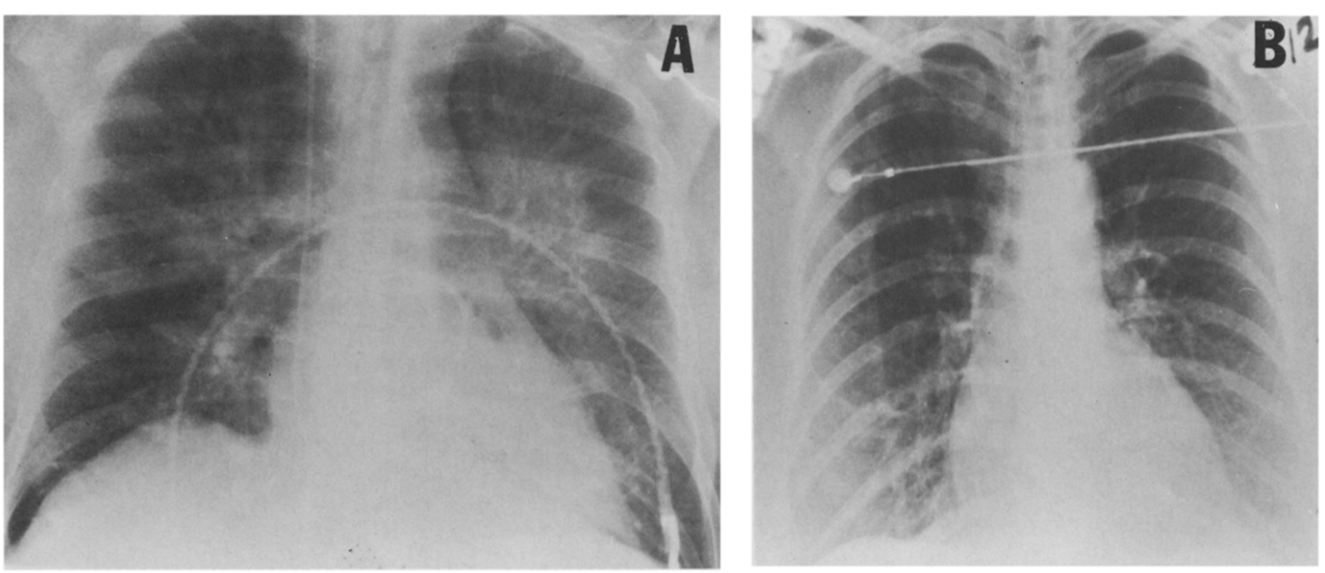


Table 2. Hemodynamic and Angiographic Findings Before and After Coronary Angioplasty in Five Patients

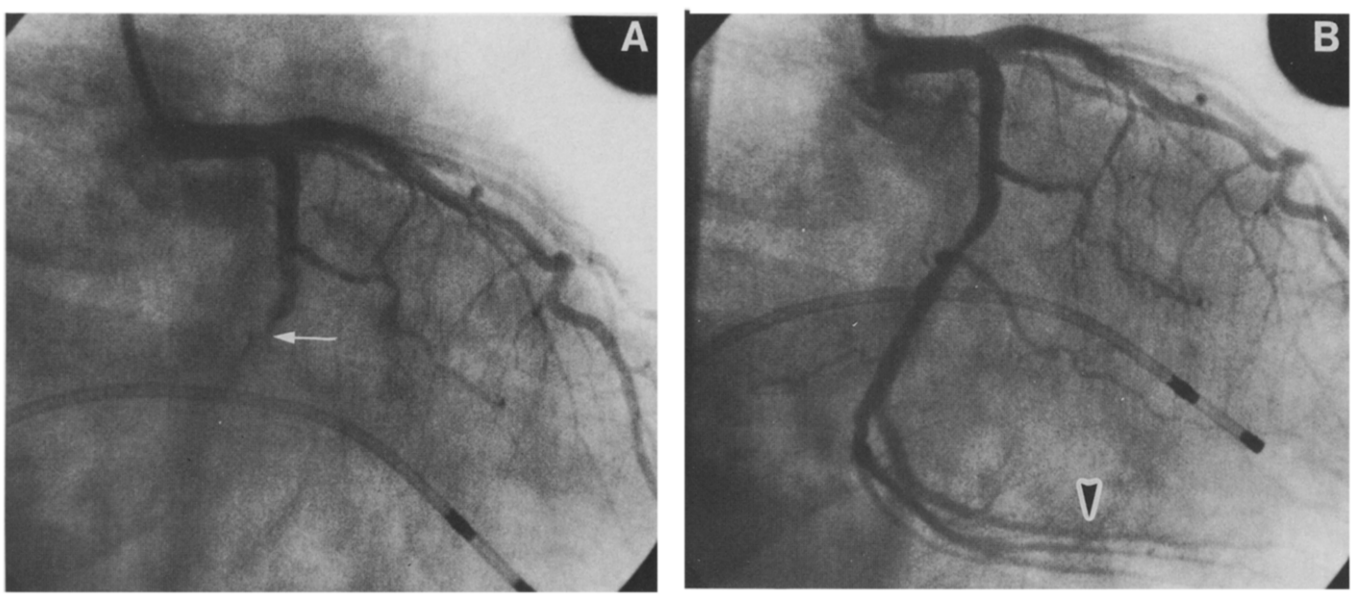
Patient No.	Coronary Arteries Narrowed (no.)	Involved Artery	Degree of Angiographic MR		% Stenosis		PA (mm Hg)		Mean PCW (mm Hg)		*V* Waves (mm Hg)		EF (%)	
			Pre	Post†	Pre	Post	Pre	Post‡	Pre	Post‡	Pre	Post‡	Pre	Post†
1	1	LCx	3+	0	100	20	60/45	30/16	32	16	50	22	48	58
2	2	RCA	4+	1+	95	20	90/45	30/22	50	18	70	26	60	62
3	2	LCx	4+	0	100	30	55/35	24/12	30	15	45	20	40	—
4	1	LCx	2+	0	100	20	56/28	20/14	26	12	38	18	58	62
5*	1	RCA	—	—	99	40	45/26	28/14	28	14	36	20	—	—

*Renal failure; †follow-up angiography was performed 4 to 6 months after angioplasty; ‡hemodynamic measurements were made 10 to 20 min after angioplasty. CA = number of coronary arteries with ≥50% luminal narrowing; EF = global left ventricular ejection fraction; LCx = left circumflex artery; MR = mitral regurgitation; PA = pulmonary artery systolic/diastolic pressure; PCW = pulmonary capillary wedge pressure; Pre = before angioplasty; Post = after angioplasty; RCA = right coronary artery.

reduction in systolic pulmonary artery pressure (from 61 ± 17 to 26 ± 4 mm Hg) and mean pulmonary capillary wedge pressure (from 33 ± 10 to 21 ± 3 mm Hg) (Fig. 3). It was possible to discontinue intraaortic balloon pulsation in the two patients soon after the procedure, and all patients were discharged within 12 days of angioplasty (mean hospital stay 10.0 ± 2.2 days). Left ventricular angiography was repeated in three patients 4 to 6 months after discharge.

Follow-up (Table 3). Follow-up study ranged from 28 to 43 months (mean 35 ± 6). Three patients underwent repeat right and left heart catheterization at 4 to 6 months (mean 5) after angioplasty. Angiographically, two patients (Patients 1 and 4) had no mitral regurgitation and one patient (Patient 2) had mild regurgitation (Fig. 4). No mitral regurgitation was noted on auscultation in the other two patients. Restenosis occurred at the site of angioplasty in two patients. Patient 4 underwent successful repeat angioplasty, and Patient 1 underwent coronary artery bypass grafting for restenosis without mitral valve replacement. Patient 2 developed severe mitral regurgitation 8 months after angioplasty without restenosis of the dilated vessel and required elective mitral valve replacement. Two-dimensional echocardiography with Doppler ultrasound was performed at a mean of 24 months after angioplasty in the other four patients, with only a trace of mitral regurgitation noted in Patient 4. All patients are alive at a mean of 35 months after angioplasty and are in functional class I.

Figure 2. Patient 3. **A**, Coronary angiogram in the right anterior oblique position showing total occlusion of the left circumflex artery after the first marginal branch (**arrow**). **B**, After angioplasty, a large posterior descending artery is visualized (**arrowhead**).



Discussion

Mechanism of papillary muscle dysfunction. Mitral regurgitation secondary to papillary muscle dysfunction is relatively common in patients with ischemic heart disease. Various degrees of mitral regurgitation have been observed

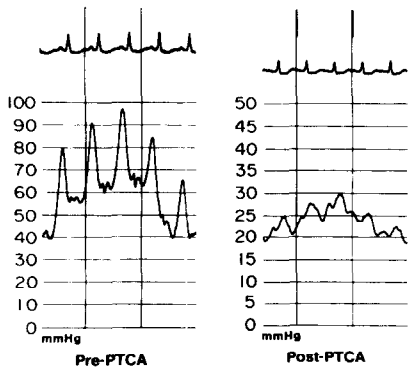


Figure 3. Patient 2. Pulmonary capillary wedge tracing before and after angioplasty (PTCA). Prominent *V* waves are present before reperfusion. After angioplasty, mean pressure is reduced and *V* waves are no longer prominent.

in 15% to 25% of patients with chronic myocardial ischemia and >50% of patients with acute myocardial infarction (3,5). The posterior papillary muscle is more prone to ischemic damage because it has a single blood supply from the posterior descending artery, which arises from a dominant right coronary or left circumflex artery. The anterior papillary muscle receives a rich blood supply from branches of the left circumflex and left anterior descending coronary arteries and, therefore, rarely develops ischemic necrosis (2,3,5).

The clinical spectrum of ischemic papillary muscle dysfunction varies from mild dysfunction producing a transient midsystolic click and murmur to severe valvular regurgitation resulting in pulmonary edema and cardiogenic shock (5,17). Brief episodes of ischemia in the experimental model (18) result in mild regurgitation due to mitral valve prolapse secondary to relative displacement of papillary muscles toward the mitral valve orifice. Persistent severe ischemia would produce necrosis of papillary muscle and the adjacent myocardium, leading to incomplete leaflet coaptation and severe regurgitation (19). Significant ischemic papillary muscle dysfunction usually is manifested in the first few hours of an evolving myocardial infarction (5). Possible sequelae include progressive regurgitation secondary to scarring of the muscle or papillary muscle rupture (4,20). Rupture usually occurs a few days after myocardial infarction and is frequently associated with cardiogenic shock and a high mortality rate (4).

Role of surgery in ischemic mitral regurgitation. Initial therapy of acute severe mitral regurgitation is directed toward stabilizing the patient's condition by decreasing myocardial oxygen requirements and reducing left ventricular afterload pharmacologically or mechanically with intraaortic balloon pulsation. Restoration of blood supply to the ischemic myocardium is obligatory to prevent irreversible necrosis of the papillary muscle. This can be achieved with thrombolytic agents, emergent coronary balloon angioplasty or surgical revascularization.

Numerous studies (6–10) have examined the role of mitral valve replacement with or without concomitant coronary artery bypass surgery in the treatment of severe mitral regurgitation secondary to ischemic papillary muscle dysfunction or rupture. The results have been generally disappointing, with mortality rates ranging from 25% to 50% (6–10). In a large series of patients with acute and chronic ischemic mitral regurgitation, Pinson et al. (6) demonstrated that the severity of valvular dysfunction and the presence of cardiogenic shock and left heart failure were the most powerful predictors of early and late death after surgery. Early deaths were observed in >70% of patients in cardiogenic shock. Percent survival was significantly reduced in patients with ischemic regurgitation compared with that in patients undergoing bypass surgery and valve replacement for nonischemic mitral incompetence. Other studies (7–10) utilizing smaller numbers of patients have shown similar results. The operative mortality rate in patients with papillary muscle rupture appears to be even higher.

Role of coronary angioplasty. Numerous studies (12–15) have demonstrated the efficacy of coronary angioplasty in the treatment of acute ischemic syndromes. Heuser et al. (21) described the successful short-term results of emergent angioplasty in three patients with acute ischemic mitral regurgitation. Angioplasty offers several advantages over

Table 3. Clinical, Angiographic and Echocardiographic Follow-Up Data in Five Patients

Patient No.	Angiographic* MR	PA* (mm Hg)	PCW* (mm Hg)	*V* Waves* (mm Hg)	Doppler Evidence† of MR	Follow-Up (months)	Comments
1	0	26/12	12	16	0	43	Restenosis, CABG
2	1+	36/24	18	24	—	38	3+ MR at 8 months, elective MVR at 12 months
3	—	—	—	—	0	34	Asymptomatic
4	0	25/12	10	14	Trace	30	Repeat PTCA
5	—	—	—	—	0	28	Asymptomatic

*Angiography was performed and hemodynamic measurements were obtained at a mean of 5 months after angioplasty; †Doppler study was performed at 24 months after angioplasty. CABG = coronary artery bypass surgery; MVR = mitral valve replacement; other abbreviations as in Tables 1 and 2.

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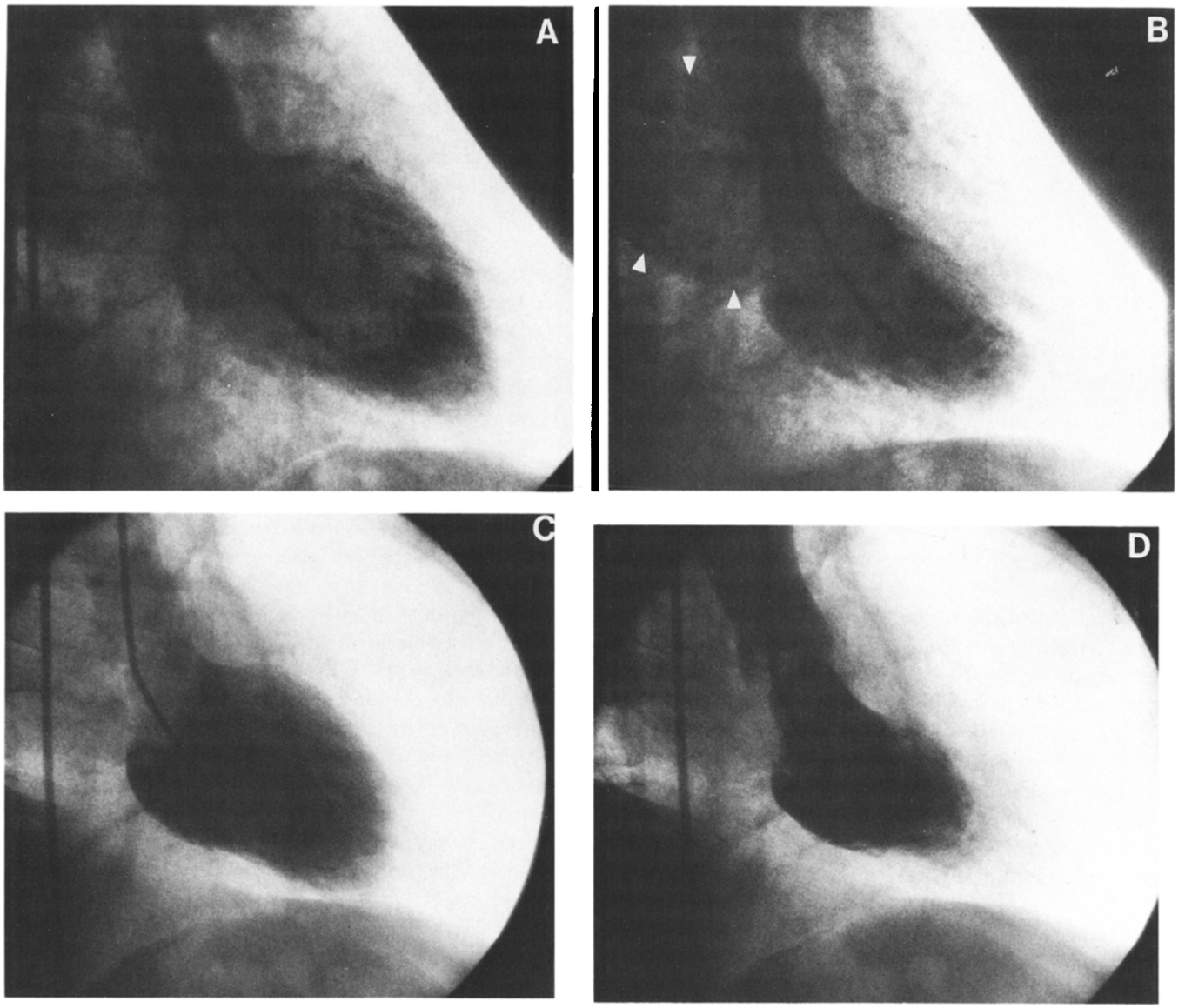

Figure 4. Patient 1. End-diastolic and end-systolic ventriculograms, respectively, in the right anterior oblique projection at presentation (**A** and **B**) and after angioplasty (**C** and **D**). The end-systolic frame (**B**) shows 3+ regurgitation into the left atrium (**arrows**); after angioplasty, no mitral regurgitation is evident (**D**).

coronary bypass surgery in patients with severe ischemic mitral regurgitation. Reperfusion can be successfully established with angioplasty in this subset of patients who have a high operative mortality risk. Because papillary muscles appear to make an important contribution to regional ventricular mechanics, preservation of papillary muscles by angioplasty may result in improved ventricular function compared with conventional mitral valve replacement (22,23). Finally, the long-term complications of bleeding secondary to anticoagulation and prosthetic valve dysfunction would also be avoided.

No studies have compared the efficacy of pharmacologic versus mechanical reperfusion in the treatment of ischemic mitral regurgitation. The advantage of thrombolytic therapy is that it may result in earlier reperfusion, particularly if initiated by paramedics before hospitalization. The initiation of intravenous thrombolytic therapy in Patient 2, who had a long period of ischemia before angioplasty, might have prevented irreversible necrosis of the posterior papillary muscle and chronic mitral regurgitation necessitating elective mitral valve replacement 12 months after angioplasty. Angioplasty would be required in patients in whom thrombolytic therapy is unsuccessful or when significant mitral regurgitation persists secondary to a severe residual stenotic lesion.

Present study. This study demonstrates the efficacy of emergent angioplasty in the treatment of severe mitral regur-

gitation secondary to ischemic posterior papillary muscle dysfunction. All patients had left ventricular failure, and two had cardiogenic shock requiring intraaortic balloon counterpulsation. Angioplasty resulted in immediate improvement in hemodynamic variables, and all patients were discharged after a mean hospital stay of 10 days. Low dose intracoronary streptokinase was utilized as adjunct therapy in three patients to lyse angiographically visible thrombus after angioplasty. All patients were alive after a mean follow-up period of 35 months, and Doppler studies in four revealed a competent mitral valve. In Patient 2, progressive mitral regurgitation without restenosis developed, necessitating elective mitral valve replacement 12 months after angioplasty. The operative findings of severe fibrosis of the posterior papillary muscle and the prolonged interval from onset of symptoms to angioplasty suggest that irreversible necrosis had already occurred in the muscle before reperfusion.

Conclusions. This study is the first to demonstrate the efficacy and long-term benefit of emergent coronary angioplasty in the treatment of ischemic mitral regurgitation. The high operative mortality rate in these patients suggests that revascularization with angioplasty or thrombolytic therapy, or both, may be the treatment of choice in this subset of patients. Further studies in a larger number of patients appear warranted to confirm these initial results.

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